

CASE REPORT

Mechanism of pacemaker induced cough

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Abstract

Single chamber ventricular pacing (VVI) may be associated with a group of adverse symptoms known as the pacemaker syndrome. Cough is an unusual but recognised feature of the pacemaker syndrome. A patient with a VVI permanent pacemaker experienced a disturbing cough during VVI pacing. There were no other symptoms associated with the pacemaker syndrome. The effects of short-term ventricular pacing on the cough were examined while the subject was standing and lying. After control recordings, the pulse generator was programmed to either VVI 50 beats/min or 90 beats/min and recordings made over 60 seconds. There was an interval of 60s between recordings. Overall, five recording periods at VVI of 50 beats/min and VVI of 90 beats/min were made in random order. The patient was blinded to the order of programming. The recordings were repeated with the subject lying. Cough was not found during normal sinus rhythm. During VVI pacing the patient experienced a tickling sensation in the throat associated with intermittent coughing. The number of coughs decreased during each successive recording period. The pacing cough reflex was enhanced when the patient was lying down. The mechanism of cough during VVI pacing is uncertain. The findings suggest a possible role for afferent vagal receptors from the airways.

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Single chamber ventricular (VVI) pacing may be associated with unpleasant symptoms collectively referred to as the pacemaker syndrome.¹ Cough is an unusual but recognised feature of the pacemaker syndrome.² We describe a patient who experienced cough as the only symptom during VVI pacing and discuss the possible neural mechanisms.

Case report

A 60 year old man was referred for assessment of recurrent syncope. The history was suggestive of Stokes-Adams attacks. Medical history was unremarkable. Examination

found a pulse rate of 65 beats/min and a blood pressure of 140/75 mm Hg. There was no evidence of heart failure. An electrocardiogram showed sinus rhythm of 60 beats/min with first degree atrioventricular block. A 24 hour ambulatory electrocardiogram was unremarkable. An echocardiogram was reported to show some thickening of the non-coronary cusp of the aortic valve with a Doppler derived gradient of 20 mm Hg. During exercise tests the patient developed partial right bundle branch block (heart rate 150 beats/min). A single chamber ventricular permanent pacemaker (Teletronics Reflex 8220) was inserted with a bipolar electrode and programmed to demand pacing at 50 beats/min.

At a routine pacing check up he reported an occasional dry, tickling cough present since pacemaker insertion. There were no other symptoms. When a magnet was placed over the pulse generator (pacing mode changed from VVI of 50 beats/min to VOO of 80 beats/min a dry cough developed that was associated with a tickling sensation at the back of his throat.

The effects of short-term ventricular pacing on cough were examined while the subject was standing erect. An electrocardiogram (lead II) and transthoracic impedance (reflecting chest wall movement) were recorded during an initial 60 second control period. The pulse generator was then reprogrammed to either VVI 50 beats/min or 90 beats/min and recordings made over 60 seconds. Between each recording period there was a 60 second control period. Overall, five VVI 50 beats/min and five VVI 90 beats/min recording periods were obtained in random order. The procedure was then repeated with the subject supine. The interval between the last recording standing and the first recording supine was 60 seconds. The order of programming was blinded to the patient.

Coughing was not found during VVI pacing at 50 beats/min—that is, during normal sinus rhythm. During VVI pacing at 90 beats/min the patient experienced a tickling sensation in the throat associated with intermittent coughing (fig 1). Figure 2 shows the number of coughs recorded during each 60 second period (VVI 90 beats/min), standing and lying. The number of coughs decreased during each successive recording interval and during the final recording periods coughing

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Figure 1 Cough during VVI pacing. Top panel shows electrocardiogram (lead II), and lower panel thoracic impedance (reflecting chest wall movement). Inspiration causes downward deflection of impedance trace. Vertical signal (I) marks the change from VVI 50 beats/min to VVI 90 beats/min. Interference on the impedance trace represents artefact from programmer. Coughing results in large amplitude biphasic deflections of impedance trace. Paper speed is 10 mm/s.

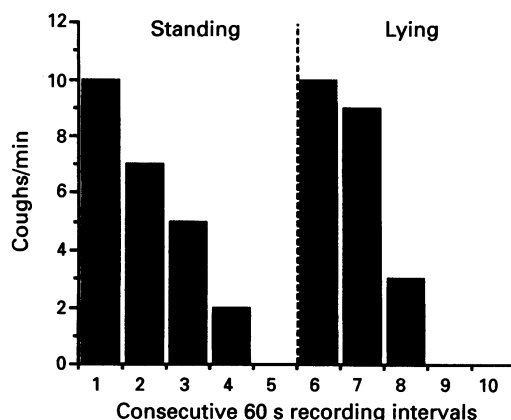


Figure 2 Effects of posture on pacemaker induced cough during VVI of 90 beats/min pacing. There is significant tachyphylaxis to repeated VVI pacing but cough response was greatly enhanced when the patient was lying down.

was absent, although the patient continued to experience a tickling sensation in the throat. When the patient was lying down, the pacing cough reflex was enhanced. During VVI pacing 90 beats/min independent atrial activity was noted on the surface electrocardiogram. Retrograde atrioventricular conduction was absent.

Discussion

We found that short-term ventricular pacing (VVI) produced coughing associated with a tickling sensation in the throat. Also, the cough response to VVI pacing showed considerable tachyphylaxis to repeated stimulation. Finally, the response to VVI pacing was considerably enhanced when the patient was lying down.

The start of acute ventricular pacing (VVI) after sinus rhythm may produce significant adverse haemodynamic changes. For example, loss of the temporal relation between atrial and ventricular contraction leads not only to loss of the atrial contribution to ventricular filling but also, in some instances, to cannon waves and mitral regurgitation,^{3,4} all of which may contrive to increase left atrial

pressure.⁵ Change from standing to lying would be expected to increase left atrial pressure further. Despite the absence of direct pressure measurements, the findings in our patient suggest that the cough associated with VVI pacing is likely to be a function of acute changes in left atrial pressure.

There are extensive communications between the bronchial and pulmonary venous systems, such that increments in left atrial pressure are likely to impair bronchial venous drainage resulting in bronchial venous congestion and oedema.⁶ These events may, in turn, be expected to influence the activity of neural elements associated with the airways.

Within the lungs and airways there are four distinct groups of vagal sensory receptor that discharge into the vagus and transmit information to the central nervous system. These include slowly adapting receptors, rapidly adapting receptors, bronchial C fibres, and pulmonary C fibres.⁷ Under specific conditions, each receptor type has been implicated in the production of cough in humans. In the experimental animal, however, the rapidly adapting receptors seem to be particularly sensitive to manoeuvres designed to promote pulmonary venous congestion. For example, an increase of left atrial pressure by a factor of only 5 mm Hg may result in a considerable increase in receptor activity. Bronchial C fibres have been implicated in the production of cough in dogs but are relatively insensitive to changes in left atrial pressure and are unlikely to be influenced by the haemodynamic changes associated with VVI pacing.⁸

We found considerable tachyphylaxis of the cough reflex during repeated periods of VVI pacing. In humans, tachyphylaxis of the cough reflex is well described after inhaled citric acid or prostaglandin E₂,^{9,10} but the coughing that follows inhaled capsaicin, the extract of red pepper, does not show adaptation.¹¹ These findings may be explained by selective receptor stimulation. Both citric acid and prostaglandin E₂ may selectively stimulate rapidly adapting receptors whereas capsaicin selectively stimulates bronchial C fibres. It may be argued then that tachyphylaxis of the

cough found during VVI pacing is determined by the precise afferent mechanism involved.

We suggest that the cough associated with the pacemaker syndrome is due to acute, but not necessarily large increases in left atrial pressure. The afferent limb of the cough reflex may be due to stimulation of the rapidly adapting receptors of the airways.

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